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ON THE DETERMINING CAUSES OF VESICULAR EMPHYSEMA OF THE LUNG.

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SICK CHILDREN, ETC.

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SINCE the attention of physicians was fixed on vesicular pulmonary emphysema by Laennec, and the anatomical character of the disease demonstrated by that pathologist, the question as to the determining cause of the dilatation of the air-cells has been a fruitful source of controversy. The question to be decided is one of much practical interest; for as vesicular emphysema is incapable of cure its prevention is of the highest importance, and to determine its cause is to determine the possibility of its prevention, and to some extent the means to be employed for its prevention.[1]

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[1] If the advocates of the theory now generally received, and which has been termed the inspiratory theory, be correct, then powerful expiratory efforts, so far as concerns the production of emphysema, are invariably to be encouraged as essentially preservative, the inspiratory efforts only are to be moderated. "Die Causalindication besteht . . . . in Vermici-

That vesicular emphysema may occur in lungs, the texture of which is otherwise perfectly healthy, is beyond doubt; we see examples of this daily in the acute vesicular emphysema of children. But then it is equally beyond doubt that in certain varieties of vesicular emphysema, *e.g.* that in which one or more vesicles at the apex or margin of the lung are greatly enlarged—the texture of the distended vesicles is constantly abnormal. Mr. Rainey[1] has rendered it highly probable that fatty degeneration of the tissue of the lung is one of the anatomical conditions on which loss of its elasticity and contractility depend. While my own observations have led me to the conclusion that the most frequent anatomical change of the lung having this result is fibrous degeneration, the consequence of the exudation of that variety of lymph which escapes from the capillaries where they are seat of slight, but long-continued congestion, in a person of tolerably healthy constitution. So common is the exudation of this variety of lymph in the congestions of old persons, that the changes of tissue which result from its presence are to be reckoned among the degenerations incident to old age. The loss of elasticity and contractility and permanent dilatation of the part which is its seat, if subjected to a distending force, is well illustrated by a reference to the large size so often attained by varicose veins when their walls are the seat of this degeneration.

The contractile power of the lung being impaired, it is evident,—that every single forcible distension of the air-cells must be followed by a certain degree of permanent dilatation; that an amount of expansion of the cells which would

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dung inspiratorischer Anstrengungen, welche die gewaltsame Ausdehnung der Luftzellen steigern können." (Canstatt's 'Specielle Pathologie und Therapie, Band ii, p. 636.) If the expiratory theory, which I advocate, be correct, then the great object of the physician, so far as concerns the prevention of emphysema, must be to moderate, in many cases at least, the violence of the expiratory efforts. Persons disposed to emphysema or affected to a slight degree with that disease must avoid occupations which necessitate expiratory efforts with a closed glottis, *e.g.*, carrying great weights, powerful pushing or pulling.

[1] 'Medico-Chirurgical Transactions,' vol. xxxi.

be instantly removed by the normal contractile power of the lung-tissue,—must, if that contractile power be seriously diminished, require only to be frequently repeated to lead to extreme vesicular emphysema. The air-cells, whether their walls be or be not healthy, can be distended, so far as we know, by one power only, viz., the pressure of air on their inner surface, but then the air may be driven against their walls, both during inspiration and during expiration. Hence two theories of the determining cause of emphysema of the lung, termed, respectively, the inspiratory and the expiratory theory.

The inspiratory theory. Atmospheric pressure may be brought to bear with undue force on the inner surface of the air-cells by the enlargement of the capacity of the thorax during inspiration being disproportionate to the capacity of the air-cells. Some portions of the lungs being from collapse or other cause diminished in volume and incapable of dilatation, then as the lungs are kept in contact with the thoracic parietes during inspiration by the pressure of the atmosphere expanding the air-cells, and so increasing the bulk of the organs, the obliteration and diminution in size of one or more air-cells necessitates (*supposing* the enlargement of the capacity of the thorax in inspiration to continue the same as with healthy lungs) abnormal expansion of the unobliterated air-cells.

This theory of the determining cause of emphysema is essentially that of Williams, Elliotson, Watson, Hasse, Rokitansky, and others. It was moulded into its present exact form by Dr. William Gairdner, and strongly supported by him in a series of very able papers published in 1850. Dr. Gairdner maintains, that the inspiratory theory admits of universal application, and that no other reasonable explanation can be offered of the mode in which the dilatation of the air-cells is effected. The views of Dr. Gairdner on this point have been adopted almost without question by the latest writers on the subject, *e.g.*, Rilliet and Barthez 'Maladies des Enfants,' t. i, p. 601; Sieveking (Jones and Sieveking's 'Pathological Anatomy,' p. 411).

Nor do I deny that in some cases a certain amount of dilatation of the air-cells is determined by inspiration.

The expiratory theory.—The object of this paper is to show that by far the most common and efficient cause of vesicular emphysema of the lung is the pressure of the air contained in the lung brought to bear on the inner surface of the air-cells by the expiratory efforts.

The expiratory theory I am about to advance is altogether different from the erroneously termed expiratory theory of Laennec.[1]

Powerful expiration is, it seems to me, infinitely the most frequent determining cause of acute vesicular emphysema and of the chronic vesicular emphysema which accompanies chronic bronchitis. It is probably the constant determining cause of the vesicular emphysema which supervenes on chronic congestion of the lungs and bronchial tubes, and on diseased heart, and of the atrophous emphysema of the aged. In like manner, expiration appears to be the invariable determining cause of vesicular emphysema whenever it is considerable in degree and general, or occupies chiefly or only the apex and border of the lung, and whenever the dilatation of one or more vesicles is extreme.

It has been asserted that during expiration every part of the lung is equally compressed, and, therefore, that the cells of no one part can be over distended. Dr. Gairdner observes:

"But the most serious objection to the expiratory theory is that the expiratory act is mechanically incapable of producing distension of the lung or of any part of it. The act of expiration tends entirely towards emptying the air-vesicles by the uniform pressure of the external parietes of the thorax upon the whole pulmonary surface; and even when the air-vesicles are maintained at their maximum or normal state of fulness by a closed glottis, any further distension of them is as much out of the question as would be the further distension of a bladder blown up and tied at the neck by hydrostatic or equalised pressure applied to its entire external surface. The air-vesicles can sustain no distending pressure from the column of air within the tubes, as that air only becomes compressed in virtue of a force which opposes exactly as much resistance without as it creates pressure within. It is singular that a theory so radically unsound and so devoid of proof should have been allowed to maintain a place in medical literature." ('Monthly Journal of Medical Science,' v. xiii, p. 10.)

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[1] Laennec's is in reality an inspiratory theory. Starting with the assertion that the inspiratory force is greater than the expiratory, he states his theory thus, "l'air, après avoir forcé la resistance que lui opposait la mucosité ou la tuméfaction de la membrane muqueuse bronchique, ne peut la vaincre dans l'expiration, et se trouve emprisonné par un mécanisme analogue à celui de la crosse du fusil à vent. Les inspiration suivantes, ou au moins les plus fortes d'entre elles amenant dans le même lieu une nouvelle quantité d'air, produisent nécessairement la dilatation des cellules aériennes auxquelles se rend la bronche oblitérée." ('Traité de l'Auscultation Médiate,' tom. i, p. 302.) As the temperature of the air in the lungs is considerably above that of the external air, its dilatation after entering the air-cells must tend still further, Laennec fancied, to over distend the air-cells.

But is it true that during expiration every part of the lung is equally compressed? Is it true that every part of the thoracic walls are during powerful expiratory efforts equally unyielding?

We have only to watch a person whose chest is exposed during a fit of coughing to see what a consideration of the anatomical constitution of the thoracic parietes would *à priori* lead one to assert, viz., that the answer to both these questions must be in the negative.

Before coughing a person makes a deep inspiration, *i. e.* he distends as far as possible the air-cells, he then closes the glottis and compresses forcibly the lungs by the thoracic and abdominal parietes, the moment the compression of the lungs has attained a certain point, he opens more or less the glottis, and the air is driven forward by the muscular effort and the elasticity and contraction of the lungs and of the thoracic walls with a force proportionate to the compression to which it was subjected before the opening of the glottis.

Now it is manifest that if there be parts of the thoracic walls which are more yielding, or which during powerful expiratory efforts with a closed glottis contract less than others, that the air immediately before the opening of the glottis will be driven from the compressed portions of the lung into the air-vesicles of the lung situated under such parts of the walls with a force proportionate to the general muscular and other powers in play, to the local want of compression and to the degree of yielding of the walls at those particular spots. That there are such parts, and that they are exactly those which are most frequently the seat of vesicular emphysema, and the sole seats of extreme dilatation of the air-cells is demonstrable.

Above the first rib the lung is covered only by soft tissues, and certainly, compared with the part of the lung situated below the level of the second rib, it is subjected during ordinary respiratory efforts to very little external compression.

The air-cells of the lung above the first rib being then surrounded by comparatively yielding walls, and being comparatively or absolutely uncompressed during expiration, ought, one would *à priori* suppose, to be distended to the utmost when powerful expiration is made with a closed or imperfectly opened glottis; the air being necessarily driven from the compressed to the comparatively uncompressed parts of the lungs. And that during violent expiration with a more or less closed glottis, the air is actually driven into the lung-vesicles of the apex with power enough to distend them to the utmost, is demonstrated by the supra-clavicular bulging which may be seen during a fit of coughing; and the hand has only to be placed upon the same part to prove that the lung tissue subjacent is, during strong expiration, distended by a considerable force. Percussion proves that the bulging to which I refer is pulmonary. If the apex of the lung be the seat of emphysema then the supra-clavicular bulging during violent expiration is extreme, and percussion on the bulging part elicits an almost tympanitic sound.[1]

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[1] The force which is exerted on the walls of the air-cells when the glottis is closed and powerful expiratory efforts made is also shown by

The cells of the anterior margin of the lung are, like those of the apex, forcibly distended when violent expiratory efforts are made with the glottis closed, or imperfectly opened. The expiration of coughing is performed chiefly by the abdominal muscles forcing up the diaphragm, the ribs compressing the lungs laterally, and the sternum and the cartilages of the ribs anteriorly. But the cartilages are to some degree yielding, and the anterior margin of the lungs is to a considerable extent protected from direct pressure by the heart and great vessels; the consequence is, that the air is driven during violent expiration from the parts most compressed (*i. e.*, the central, basic, and lateral parts), into the air-cells of the anterior margin, as well as into the air-cells of the apex.

Again, the margin of the base of the lung, the part of the lung in the vicinity of the root of the organ below the entrance of the bronchus, and the little ridge of lung which lies behind the trachea on the right side forming the posterior margin of what may be termed the tracheal groove on the lung, are in a like manner imperfectly supported and comparatively uncompressed during violent expiration. The base, too, of the left lung generally, is less firmly supported than is the corresponding part of the right lung—the liver being a more unyielding organ than the stomach, and compressing the base of the right lung more uniformly than the spleen and stomach do that of the left lung. The consequence of this want of support and of compression of the parts of the lung last enumerated is, that they, like the apex and the anterior margin, are the chosen seats of

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the fact that during the expulsive efforts in parturition one or more air-cells occasionally give way, and the air finds its way through the posterior mediastinum into the cellular tissue of the neck and face. A case of this kind fell under my observation when a student. The woman, after a violent detrusive effort, in which necessarily the glottis was closed and the muscles of expiration called into action, suddenly exclaimed, "I am unable to see." Her face and neck were, I now found, the seat of subcutaneous emphysema.

emphysema;[1] the air contained in the parts of the lung most powerfully compressed during expiration with a closed glottis, being forced in undue quantity into such parts of the lung as are less compressed and less perfectly supported.

It is manifest that what has been above advanced, as to the effect on the air-cells of powerful expiratory efforts made when the glottis is partially closed, is equally true of the same efforts when made with one or more bronchial tubes partially closed. Dilatation of some of the air-cells beyond the tubes must ensue, if those cells be not uniformly compressed during expiration.

While, therefore, collapse of the lung is the consequence of complete occlusion of a bronchial tube, dilatation of the air-cells may be the result of imperfect occlusion.

The objection to the theory of Laennec, urged by Louis, viz., that the ordinary seat of catarrh is the base of the lung posteriorly, that of emphysema the apex and anterior margin, is unanswerable. The objection of Dr. Gairdner to the same theory, viz., that impermeability of a bronchial tube is followed by collapse of the pulmonary tissue from which it leads, is equally fatal to it. But then the conclusion of Louis from his facts, viz., that there is no relation between catarrh and vesicular emphysema by no means necessarily follows. The relation between the two is, that catarrh excites frequently repeated violent expiratory efforts, with a more or less perfectly closed glottis, and therefore is one of the exciting causes of repeated over-distension of these air-cells, which during these efforts are the least supported and compressed. Catarrh, then, of the base of the lung posteriorly, may be the exciting cause of vesicular emphysema of the apex and anterior margin, and Louis's conclusion as to the necessary want of relation between catarrh and vesicular emphysema, because they affect different parts of the lung, is manifestly illogical.

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[1] Louis found the lower lobe of the left lung twice as often the seat of emphysema as the corresponding lobe of the right lung; while the apex of the right lung was more frequently affected with emphysema than the apex of the left lung.

Dr. Gairdner's conclusion, viz., that obstruction of a bronchus, because it is sometimes followed by collapse of the air-cells, cannot be a determining cause of rarefaction of the part of the lung from which it leads, is equally untenable. For incomplete occlusion of the bronchi may have, and in some cases I believe does have, a result the reverse of perfect occlusion, and in catarrh and bronchitis imperfect occlusion of the tubes is the rule, perfect occlusion the exception.

The notes of three cases will suffice to illustrate the points dwelt on in this paper—the facts are so common and so patent to every observer, that it would be useless to give more.

Mrs. L—, æt. 63, tall, thin; suffered from winter cough; was "asthmatical on exertion;" the chest was small in circumference, but long; the intercostal spaces were wide, and fell in during ordinary inspiration; there was some recession of the supra-clavicular regions during inspiration. On coughing there was great bulging of the supra-clavicular regions, especially the right. On percussion, the bulging part gave out an almost tympanitic sound. Two days after the foregoing observation the lady died suddenly of fatty degeneration of the heart. The apices of the lungs were the seat of extreme local emphysema.

H. C—, æt. 46, a patient under my care in University College Hospital, suffering from dry pleurisy and emphysema. The subjoined note was taken a day or two after admission. The cough is more troublesome, and there is a large amount of sonorous râle, audible during expiration, over both sides of the chest anteriorly. Below the prominence opposite the second costal cartilage the sternum recedes during each inspiration; in the act of coughing it advances with each expiratory succession; there is marked depression of the supra-clavicular regions during each inspiration, and marked bulging during each expiration. The infra-clavicular regions also advance during the expiration of coughing. The expiration of coughing is chiefly effected by the abdominal muscles, and these are called into play even as low as the pubes.

Catherine C—, æt. 1 year 9 months, was admitted under my care into the Hospital for sick children suffering from Croup. The following note was taken in the morning as she died in the evening.

Respirations 36, noisy; inspiratory and expiratory sound, as heard by the bedside, husky, hoarse. Expiration much longer than inspiration; cough frequent, hoarse, croupy, singing. During inspiration there is recession of the supra-clavicular, infra-clavicular, and supra-sternal regions of the sternum and costal cartilages below the second, and of all the intercostal spaces. During expiration there is bulging of the supra-clavicular region, and prominence of the infra-clavicular and upper sternal regions. No physical signs of bronchitis. The child died the same evening; and on examination of the body, there was found, in addition to the false membrane in the larynx and trachea, vesicular emphysema of the anterior margin of both lungs, especially strongly marked in the parts overlapping the heart and great vessels, and at the apices. There was scarcely a trace of collapse of any part of the right lung, but several small patches of collapsed tissue in the inferior lobe of the left lung. The emphysema was as great of the right as of the left lung.

During the cough, which takes place in a fit of hooping-cough, the same supra-clavicular bulging, advance of the sternum, &c., as were observed in the above case, are well seen, as are, during the hoop, the recession of the supra-clavicular region, of supra-sternal region, &c.

I may sum up the position I have taken in regard of the question as to the determining cause of vesicular emphysema of the lung, thus:

1. The lung during expiration is compressed at different parts with different degrees of force.

2. The parietes of the thorax, in consequence of their

anatomical constitution, yield to the same force at different parts with various degrees of facility.

3. The chosen seats of emphysema are exactly those parts of the lung which are the least compressed during expiration, and the least affected by inspiration, and which are situated under those portions of the thoracic parietes that give way the most readily before pressure.

4. The power possessed by man of dilating the apex of the lung by inspiratory effort is altogether insufficient to distend the air-cells to the degree to which they are often found to be distended in local emphysema of the apex.

5. That the air-cells of the apex can be distended very considerably and with force during expiration is matter of demonstration; for in violent coughing we may see the elevation of the supra-clavicular region, the advance of the upper part of the sternum, and of the intercostal spaces generally during each expiratory succussion, the bulging parts being resonant on percussion.

6. When an impediment exists to the entrance of the air into the lungs and to its egress from the lungs, as in croup and in hooping-cough, the thoracic walls over the parts of the lungs, which after death are found emphysematous, recede during inspiration and advance during expiration.

7. In the cases last mentioned the more violent the inspiratory efforts the greater the recession of the parts of the parietes referred to, and the more violent the expiratory efforts the greater their advance.

8. When the apex is the seat of calcified or of obsolescent tubercle, lymph is exuded around and into all the tissues of the apex. Some air-cells are obliterated and the elasticity and contractility of the tissues generally is either diminished or lost.

9. Under the circumstances last specified dilatation of the unobliterated cells by the air driven into them during violent expiration with a closed glottis attains its maximum.

10. Under the same conditions in the same situation, the inspiratory force is altogether insufficient to cause dilatation of the air-cells.

11. The atmospheric air moved by the inspiratory effort can exert comparatively little pressure on the inner surface of the air-cells situated at the extreme margin of the base, the root of the lower lobe (*i. e.* that part immediately next the spine and below the primary bronchus) or at the part of the apex situated in the furrow posterior to the trachea on the right side. While violent expiration, being chiefly performed or greatly aided by the abdominal muscles forcing upwards the liver, &c., drives the air (in consequence of the highly arched form of the diaphragm in violent expiration) from the central part of the lung, not only through the bronchi towards the larynx, but also towards the circumference of the lungs, *i. e.*, towards those parts which are the least compressed during expiration.

12. In the parts just named dilatation of the air-cells is extremely common, and the dilated cells are often of large size.

13. While complete obstruction of a bronchial tube is followed by collapse of the pulmonary tissue on its distal side; partial closure of a tube has as its consequence dilatation of some of the air-cells of the part of the lung from which it leads, provided that part be unequally supported and compressed during expiration.

14. Vesicular emphysema is the invariable and necessary consequence of whatever impedes the free exit of air from the lungs, and at the same time excites powerful expiratory efforts; because by such efforts the air is driven from the more powerfully compressed parts of the lungs into those parts which are less powerfully compressed and the walls over which yield more or less readily to pressure.

Postscript.—If the view of the determining cause of emphysema advanced in this paper be correct, then in the lower animals subject to pulmonary vesicular emphysema the disease ought to be found to affect especially those parts of the lungs which in them are, during expiratory efforts with a closed glottis, the least compressed, and which are situated under those parts of the thoracic walls that are the least capable of offering resistance to pressure. To ascertain whether it is so or not, I examined, a day or two since, with the assistance of Mr. Henderson, the veterinary surgeon of Great Scotland Yard, five horses, the lungs of which were emphysematous. In four of the five horses the anterior lobes only, and that part of the inferior lobe of the right lung which is protected by the trachea were affected; in the fifth, in addition to these parts, the margin of the inferior lobe.

Now the anterior lobe of the lung in the horse is situated above and over the heart, under the four upper ribs. It is placed, that is to say, in a part of the thorax where it can during expiration be subjected to very little external compression. So the part of the right lung, which in all five horses was emphysematous, was protected from compression by the trachea. In the lungs which were the most emphysematous there was no evidence of other disease, so far as I could judge, no trace of recent or of old collapse.

The frequency with which horses suffer from emphysema is of course attributable to their being so often compelled to make (in pulling, &c.) powerful expiratory efforts with a closed glottis. One would expect to find all animals (man among the others), engaged in occupations necessitating continued and repeated expiratory efforts with a closed glottis, more subject to pulmonary vesicular emphysema than others of the same class, *e.g.*, draught oxen rather than milch cows.